Gestational Diabetes: The Emperor Has No Clothes
by Henci Goer

Good medicine demands that diagnosis and treatment of any disease fulfill four criteria:

- The condition has to pose a health risk;
- Diagnosis must accurately distinguish between those who have the disease and those who don't;
- Treatment should be effective; and
- The benefits of diagnosis and treatment should outweigh the risks.

An entire medical industry has grown up around diagnosing and treating gestational diabetes (GD) in the belief that doing so prevents perinatal deaths, congenital anomalies, neonatal complications, macrosomic babies, and because of fetal macrosomia, birth injuries and excessive cesarean rates. However, diagnosis and treatment of gestational diabetes don't fulfill any of the above criteria.

To begin with, GD doesn't fit the definition of a disease. GD as a concept began in 1964 when O'Sullivan and Mahan performed a 100g 3- hour oral glucose tolerance test (OGTT) on 752 pregnant women and tracked all women with at least two values above two standard deviations beyond the mean to see if hyperglycemic women were predisposed to develop diabetes down the road (O'Sullivan 1964). They were, leading the two researchers to conclude that the metabolic stress of pregnancy revealed a woman's "pre-diabetic status." This should not surprise anyone since overweight women are more likely to have hyperglycemia in pregnancy and to develop diabetes later in life.

Since insulin-dependent diabetes was known to threaten the fetus, researchers extrapolated that sub-diabetic glucose elevations might also do harm. This leap in logic was faulty on its face because GD does not share the risk factors of either type of true diabetes. In Type I diabetes, extremes of low and high blood glucose early in pregnancy can cause congenital anomalies or kill the forming embryo. Gestationally diabetic women make normal or above-normal amounts of insulin and have normal blood sugar metabolism in the first trimester. With either Types I or II, diabetes of long standing may damage maternal blood vessels and kidneys, causing hypertension or kidney complications. These may in turn jeopardize the fetus. Gestational diabetics do not have long- standing diabetes. The one problem GD shares with both types is that chronic hyperglycemia can overfeed the fetus, resulting in macrosomia (generally defined as birth weight greater than 4000 g) or large-for- gestational-age (LGA) (greater than the 90th percentile) babies.

Logic notwithstanding, these concerns launched a series of studies into the risks of mild glucose elevations. Unfortunately, they were badly flawed.

- Studies selected women for glucose testing based on such factors as prior still birth or hypertension in the current pregnancy and then compared outcomes with the general population. Hunter and Keirse observe that according to Sutherland and Stowers' 1975 edition of CARBOHYDRATE METABOLISM IN PREGNANCY AND THE

NEWBORN, the rate of fetal loss increases eightfold as the number of indications for glucose tolerance testing increasing from one to four. Glucose intolerance does not add to this risk (Hunter and Keirse 1989).

- Studies included women who were known diabetics prior to pregnancy.
- Studies failed to account for confounding factors such as that glucose intolerance associates with increasing maternal weight and age, which themselves are strong independent predictors of macrosomia and maternal hypertension.
- Studies used management protocols that increased risks such as starvation diets, early elective induction, and withholding nourishment from the newborn.

In addition, glucose level turned out to be a poor predictor of macrosomia. Other factors such as race, age, parity, sex, and especially maternal weight, far outweighed glucose intolerance in determining birth weight. Hunter and Keirse observed that GD mothers had a 3-fold risk of giving birth to a baby weighing over 4500 g compared with normoglycemic women. However, a woman weighing over 90 kg had a 26-fold risk of having a baby this heavy compared with normal weight women (Hunter and Keirse 1989). Oats and colleagues could not find a significant association between glucose levels and birth weight until birth weight exceeded the 90th percentile. Even then, 77 percent of women had normal glucose tolerance (Oats et al. 1980).

Nonetheless, researchers concluded that mildly deviant glucose values in pregnancy constituted a new form of diabetes that required diagnosis, surveillance, and treatment. Researchers have gone on adding rooms and stories to the GD edifice, never noticing that they have built a house on sand.

Secondly, the OGTT, the standard diagnostic test, has many problems. A diagnostic test should be reproducible, its thresholds should be values at which morbidity either first appears or incidence greatly increases, and normal ranges should apply to the population undergoing testing. The OGTT is none of the above.

Obstetricians adopted O'Sullivan and Mahan's curve as the normative curve for all pregnant women, but it is not representative. For one thing, O'Sullivan and Mahan tested women without regard to length of gestation, whereas today, women are typically tested at the beginning of the third trimester. Glucose values rise linearly throughout pregnancy, but no corrections have been made for this. For another thing, O'Sullivan and Mahan studied a population that was 60 percent white and 40 percent black. Hispanics, Native Americans, and Asian women average higher blood sugars than black or white women. Since diagnostic thresholds are set at two standard deviations beyond the mean, values for O'Sullivan and Mahan's population have arbitrarily been established as the norms for all women. This means that some women are being identified as diseased simply because of race.

Worse yet, studies show that when pregnant women undergo two OGTTs a week or so apart, test results disagree 22 percent to 24 percent of the time (Catalano et al. 1993) (Harlass et al. 1991). An individual's blood sugar values after ingesting glucose (or food) vary widely depending on many factors. For this reason, the OGTT has been abandoned as a diagnostic test for true diabetes in favor of excessive fasting glucose values, which show much greater consistency, or postprandial values of 200 mg.dl or more, which are rare. Moreover, pregnancy compounds

problems with reproducibility. Because glucose levels rise linearly throughout pregnancy, a woman could "pass" a test in gestational week 24 and "fail" it in week 28. These same problems hold true for the glucose screening test that precedes the OGTT (Sacks et al. 1989) (Watson 1989).

More importantly, no threshold has ever been demonstrated for onset or marked increase in fetal complications below levels diagnostic of true diabetes. O'Sullivan and Mahan chose their cutoffs for convenience in follow-up, but all studies since then have used their criteria or some modification thereof as a threshold for pathology in the current pregnancy. Numerous studies since have documented that birth weights and other outcomes fail to correlate with O'Sullivan's or anybody else's thresholds.

A test with arbitrary diagnostic thresholds is akin to claiming that all people over six feet tall have a growth abnormality or all people with a cough and a fever have pneumonia. The authors of A GUIDE TO EFFECTIVE CARE IN PREGNANCY AND CHILDBIRTH relegate "screening for gestational diabetes" to "Forms of Care Unlikely to be Beneficial" (Enkin 1995).

The original intent of treating GD was preventing excess perinatal mortality and congenital anomalies. Whatever the cause of increased deaths, it wasn't hyperglycemia. O'Sullivan and colleagues randomly assigned gestational diabetics to treatment with diet and insulin and compared outcomes among treated diabetics, untreated diabetics, and a normoglycemic control population. They found more perinatal deaths in the GD population, treated or not (O'Sullivan et al. 1966). Perinatal mortality statistics among non-insulin dependent diabetics remained unchanged between 1946 and 1972 in a Copenhagen study despite aggressive treatment throughout the timespan (Pedersen, JL et al. 1974) (Pedersen J 1977). Conversely, a Swedish study showed a marked reduction in perinatal mortality rates between 1961 and 1971, also while treating vigorously (Karlsson et al. 1972).

As for congenital anomalies, GD cannot cause congenital anomalies because glucose metabolism is normal in the first trimester. Even if it did, testing isn't done until the third trimester.

The main rationale for current GD management is to reduce the incidence of birth injuries and cesarean section by reducing the incidence of macrosomia. The goal of reducing birth weight raises philosophical problems. As with glucose values, doctors are defining deviation beyond an arbitrary point as inherently pathological. Moreover, can we justify manipulating the growth mechanism of a group of babies roughly 75 percent to 80 percent of whom will fall below the 90th percentile for weight if left alone?

Philosophical considerations aside, we have little evidence that GD management succeeds. As mentioned above, macrosomia associates with maternal weight, age, race, parity, and male fetus. Maternal overweight cannot be rectified during pregnancy; the rest cannot be altered at all. According to M.J. Stephenson, there have been only four randomized trials of diet or diet and insulin. All were flawed and taken together achieved a reduction in birth weight of 87 g, a benefit "of questionable clinical significance" (Stephenson 1993). A GUIDE TO EFFECTIVE CARE IN PREGNANCY AND CHILDBIRTH also lists insulin and diet therapy for GD under "Forms of Care Unlikely to be Beneficial."

Santini and Ales report results from a national trial that occurred in the early 1980's when some doctors at Cornell University Medical Center screened women for GD routinely and others did not. No differences in perinatal mortality, morbidity, LGA or macrosomia rates were found between screened and unscreened populations, but women in the screened population were more likely to have primary cesarean sections (19 percent versus 12 percent), more clinic visits, more fetal surveillance tests, and more prenatal hospitalization (Santini et al. 1990).

Non-randomized trials show that diet modification rarely works without severely limiting calories or the liberal or universal use of insulin. Even where it does work, only two studies of GD management reduced operative delivery or cesarean rates to reasonable levels, the main point of preventing macrosomia (Langer et al. 1994) (Coustan et al. 1984). In both studies, doctors knew which women were treated and which were controls. If they believed their therapy prevented macrosomia, which other work shows they did, this belief could well have influenced management decisions. A third study also reported similar cesarean rates in GD women and the total hospital population, but these were 27 percent and 25 percent respectively (Thompson et al. 1994).

As Santini and Ales' study suggests, not only does GD management offer little benefit, it confers risks, the gravest being a marked increase in cesarean section. The cesarean rate in a population of gestational diabetics cared for by midwives was 9 percent to 11 percent including women transferred to obstetric management, or about half the primary cesarean rate reported in populations managed by obstetricians in the same or an earlier time period (O'Brien et al. 1987). Goldman and colleagues reported that gestational diabetics had one-third more cesareans compared with a matched population with normal glucose tolerance, although birth weights were similar (Goldman et al. 1991). In another study, gestational diabetics were randomly assigned to insulin or standard treatment in the third trimester in an effort to minimize macrosomia. Insulin reduced LGA rates to 13 percent compared with LGA rates of 45 percent in the diet group and 38 percent in the group that refused randomization. Despite this, cesarean rates were 14 percent and 21 percent in the diet-treated groups versus 43 percent in the insulin-treated group, a difference attributed to transferring women on insulin to the high-risk service (Buchanan 1994).

Many doctors view high cesarean rates as a reasonable trade-off for preventing shoulder dystocia. This ignores that many shoulder dystocias occur in non-macrosomic infants (Keller 1991) and that the increase in cesarean rate for infants weighing over 4000 g has not improved outcomes (Boyd et al. 1983); not to mention the role typical obstetric management plays in causing shoulder dystocia.

Increased likelihood of cesarean is not the only risk of GD management. Insulin increases the risk of small-for-gestational-age babies and causes symptomatic hypoglycemic episodes (Langer et al. 1994) (Buchanan et al. 1994). Reducing calories by more than one-third in overweight gestational diabetics causes ketosis (Knopp et al. 1991). Finally, the poor predictability of the fetal weight estimates and surveillance tests doctors feel obliged to order, even the belief that GD is a high-risk condition, undoubtedly lead to countless unnecessary inductions and operative deliveries.

Few have noticed that the diagnosis and treatment of GD is a spectacular failure. A review article analyzes the OGTT, finds it worthless, and recommends continuing to use it to diagnose GD (Nelson 1988). After showing that current cutoffs fail to discriminate a group of women at high risk for macrosomia, obstetricians conclude in defiance of logic that they should lower the values or that insulin should be given to more women or that cutoffs should be chosen by fiat (Sacks et al. 1995) (Neiger et al. 1991) (Weiner 1988) (Tallarigo et al. 1986). Researchers take note that sonography to estimate fetal weight did no better than a coin toss at predicting macrosomia and recommended it anyway (Combs et al. 1993). Doctors find that rigid glycemic control did not improve infant outcomes and assume that means they should try harder (Hod et al. 1980). Goldman and colleagues, with similar birth weights but one-third more cesareans in the GD group, congratulated themselves on the success of their management (Goldman et al. 1991). The gestational diabetes literature reads more like ALICE IN WONDERLAND than science.

Still, midwives can winnow some grain from the chaff. Maternal weight has the strongest correlation with macrosomia rate; it makes sense to advise heavily overweight women to lose weight before becoming pregnant. Pregnancy makes extra demands on insulin production; to minimize the pressure, pregnant women should eat a diet low in simple sugars, high in complex carbohydrates and fiber, and moderate in fat. Moderate, regular exercise also improves glucose tolerance. Within the GD population lurk a few women who were either undiagnosed pregestational diabetics or who were tipped into true diabetes by the metabolic stress of pregnancy; a fasting glucose to screen for them might be prudent. And, of course, midwives already use strategies that help women minimize the likelihood of operative delivery or birth injury. Finally, to reduce the chance of neonatal hypoglycemia, the baby should be put to breast soon after the birth, especially if the baby is big, small, or the labor has been difficult.

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Bibliography

- Goer, H. OBSTETRIC MYTHS VERSUS RESEARCH REALITIES: A GUIDE TO THE MEDICAL LITERATURE. Westport: Bergin and Garvey, 1995.
- "Gestational diabetes," INTERNATIONAL JOURNAL OF CHILDBIRTH EDUCATION. 1991;6(4):1991.
- "Gestational diabetes: It's Not What It Seems," CHILDBIRTH INSTRUCTOR. In press

1. O'Sullivan JB. and Mahan CM. Criteria for the oral glucose tolerance test in pregnancy. DIABETES 1964;13:278-285. [Ed. - No Abstract Available]

2. Hunter JS. and Keirse MJNC. GESTATIONAL DIABETES. In EFFECTIVE CARE IN PREGNANCY AND CHILDBIRTH, Enkin M. Keirse MJNC, and Chalmers, eds. Oxford: Oxford University Press, 1989.
3. Oats JN. et al. Maternal glucose tolerance during pregnancy with excessive size infants. OBSTET GYNECOL 1980;55:184-186.
4. Catalano PM, et al. Reproducibility of the oral glucose tolerance test in pregnant women. AM J OBSTET GYNECOL 1993;169(4):874-881.
5. Harlass FE. Brady K. Read JA. Reproducibility of the oral glucose tolerance test in pregnancy. AM J OBSTET GYNECOL 1991;164(2):564-568.
6. Sacks DA. et al. How reliable is the fifty-gram, one-hour glucose screening test? AM J OBSTET GYNECOL 1989;161(3):642-645.
7. Watson WJ. Serial changes in the 50-g oral glucose test in pregnancy: implications for screening. OBSTET GYNECOL 1989;74(1):40-43.
8. Enkin M. et al. A GUIDE TO EFFECTIVE CARE IN PREGNANCY AND CHILDBIRTH, 2nd ed. Oxford: Oxford University Press, 1995.
9. O'Sullivan et al. The potential diabetic and her treatment in pregnancy. OBSTET GYNECOL 1966;27:683-689. [Ed. - No Abstract Available]
10. Pedersen JL. Molsted-Pedersen, and Andersen B. Assessors of fetal perinatal mortality in diabetic pregnancy: analysis of 1,322 pregnancies in the Copenhagen series 1946-1972. DIABETES 1974;23:302-305. [Ed. - No Abstract Available]
11. Pedersen J. "White Class-A-mild diabetes in pregnancy" and "Management of diabetic pregnancy and the newborn infant," in THE PREGNANT DIABETIC AND HER NEWBORN, 2nd ed. Baltimore: the Williams and Wilkins Co., 1977.
12. Karlsson K. and Kjellmer I. The outcome of diabetic pregnancies in relation to the mother's blood sugar level. AM J OBSTET GYNECOL 1972;112:213-220. [Ed. - No Abstract Available]
13. Stephenson MJ. Screening for gestational diabetes mellitus: a critical review. J FAM PRACT 1993;37(3):27-283.
14. Santini DL. and Ales KL. The impact of universal screening for gestational glucose intolerance on outcome of pregnancy. SURG GYNECOL OBSTET 1990;170(5):427-436.
15. Langer O. et al. Intensified versus conventional management of gestational diabetes. AM J OBSTET GYNECOL 1994;170(4):1036-1047.
16. Coustan DR. and Imarah J. Prophylactic insulin treatment of gestational diabetes reduces the incidence of macrosomia, operative delivery, and birth trauma. AM J OBSTET GYNECOL 1984;150(7):836-842.
17. Thompson DM. et al. Tight glucose control results in normal perinatal outcome in 150 patients with gestational diabetes. OBSTET GYNECOL 1994;83(3):362-365.
18. O'Brien ME. and Gilson G. Detection and management of gestational diabetes in an out-of-hospital birth center. J NURSE-MIDWIFERY 1987 Mar/Apr;32(2):79-84. [Ed. - No Abstract Available]
19. Goldman M. et al. Obstetric complications with GDM. Effects of maternal weight. DIABETES 1991;40(Suppl 2):79-82.
20. Buchanan TA. et al. Use of fetal ultrasound to select metabolic therapy for pregnancies complicated by mild gestational diabetes. DIABETES CARE 1994;17(4):275-283.

21. Keller JD. et al. Shoulder dystocia and birth trauma in gestational diabetes: a five-year experience. AM J OBSTET GYNECOL 1991;165(4 Pt 1)928-930.
22. Boyd ME, Usher RH, and McLean FH. Fetal macrosomia: prediction, risks, proposed management. OBSTET GYNECOL 1983;61(6):715-722.
23. Knopp RH. et al. Metabolic effects of hypocaloric diets in management of gestational diabetes. DIABETES 1991;40(Suppl 2):165-171.
24. Nelson RL. Oral glucose tolerance test: indications and limitations. MAYO CLIN PROC 1988;63(3):263-269.
25. Sacks DA. et al. Toward universal criteria for gestational diabetes: the 75-gram glucose tolerance test in pregnancy. AM J OBSTET GYNECOL 1995;172:607-614.
26. Neiger R and Coustan DR. Are the current ACOG glucose tolerance test criteria sensitive enough? OBSTET GYNECOL 1991;78(6):1117-1120.
27. Weiner CP. Effect of varying degrees of "normal" glucose metabolism on maternal and perinatal outcome. AM J OBSTET GYNECOL 1988;159(4):862- 870.
28. Tallarigo L et al. Relation of glucose intolerance to complications of pregnancy in nondiabetic women. NEW ENGL J MED 1986;315(16):989-992.
29. Combs CA. Singh NB, and Khoury JC. Elective induction versus spontaneous labor after sonographic diagnosis of fetal macrosomia. OBSTET GYNECOL 1993;81(4):492-496.
30. Hod M. et al. Gestational diabetes mellitus: a survey of perinatal complications in the 1980's. DIABETES 1991;40(Suppl 2):74-78.